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Can mycophenolate mofetil be an effective option in the treatment of vascular Behçet's Syndrome

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Abstract. *Background/Aim:* Behcet's syndrome (BS) is a type of vasculitis that primarily affects veins and tend to involve pulmonary arteries more often than peripheral arteries or the aorta. Inflammation is a central role in the development of thrombotic events in BS. Given its critical role in etiopathogenesis, the use of immunosuppressive therapy is fundamental to the treatment strategy. The aim of our study is to report our center's experience with mycophenolate mofetil (MMF) treatment in vascular BS (VBS) patients. *Materials and Methods:* The clinical, laboratory and imaging findings of the patients receiving MMF treatment for vascular BS were retrospectively evaluated. It was noted whether MMF treatment was induction or maintenance treatment. Treatment related side effects were noted. On the 12th month, patients were evaluated for the development of new events and being in remission. *Results:* Five patients underwent MMF treatment for remission induction, while another five patients received MMF for maintenance. Relapses occurred in 2 out of 5 patients who were administered MMF as induction therapy, resulting in the development of acute deep vein thrombosis. Among the five patients receiving MMF as maintenance treatment, only one exhibited an active vascular event. Additionally, one patient was classified as vascularly active based on a CRP level of 10.3 mg/l. *Conclusion:* MMF therapy may be an effective and safe treatment agent that can be preferred as azathioprine and cyclosporine in VBS patients, especially in maintenance therapy. This study is the first investigation into MMF treatment in patients with vascular BS.

Key words: Behcet syndrome, mycophenolate mofetil, vascular involvement, immunosuppressive treatment

Introduction

Behçet's syndrome (BS) is a chronic inflammatory disease characterized by mucocutaneous manifestations, including oral aphthae and genital ulcers, as well as the potential involvement of major organs and systems, such as the neurological and gastrointestinal systems, and the eyes. It can also impact vessels of varying sizes, including small, medium, and large sizes (1, 2). BS predominantly affects veins and shows a higher incidence of involvement in pulmonary arteries, which share structural similarities with veins, compared to peripheral arteries and the aorta. This vasculitis is characterized by a significant predisposition to thrombosis in the absence of thrombophilia (3-6). It frequently presents as deep vein thrombosis (DVT). An aneurysm or thrombosis may occur in the pulmonary artery. Rarely, it can lead to aneurysm or thrombotic occlusion in the peripheral artery (4-6). The prevalence of vascular involvement in BS has been reported to be 40–50% (7, 8). Inflammation is a significant factor in the development of thrombotic events in BS. Thrombo-inflammation resulting from neutrophil-triggered inflammation is a mechanism that is particularly highlighted. The role of inflammation in the etiopathogenesis, immunosuppressives are the key in the treatment strategy (9-12).

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Immunosuppressive agents have been used for many years; however, there is a scarcity of large-scale randomized controlled studies specifically addressing vascular involvement. The European Alliance of Associations for Rheumatology (EULAR) recommends the use of immunosuppressive treatments, such as azathioprine (AZA), cyclosporine A (CsA), and cyclophosphamide, combined with glucocorticoids (GCs) for those with venous involvement. If the patient is resistant to this treatment, monoclonal TNF-inhibitors (TNFi) are recommended. Treatment with cyclophosphamide or monoclonal TNFi combined with GCs is recommended for conditions with more severe manifestations, such as arterial involvement. The latest EULAR guidelines make no mention of mycophenolate mofetil in the treatment of vascular involvement (13). Although there are recommendations and retrospective studies on the use of AZA and CsA in vascular BS (VBS), there are no studies showing the efficacy of mycophenolate mofetil (MMF) in cases of VBS (14, 15) . The only randomized controlled trial showing that AZA is effective in VBS is the extension phase of the study by Hamuryudan et al. in patients with ocular BS, which reported less vascular involvement in patients receiving AZA (16, 17).

Considering the potential side effects of AZA and CysA use, there is also a need for a third oral immunosuppressive such as MMF in VBS patients. Although newer drugs are being developed, additional immunosuppressive drugs may be needed in some patients due to reasons such as the presence of malignancy and refusal of biological drug use. MMF is a commonly used and effective treatment agent for small vessel vasculitis. Although there are data on ocular and neurological involvement in BS, there is no record of vascular involvement(18-20).

The aim of this study is to evaluate the clinical effectiveness and potential side effects of MMF treatment in VBS patients.

Method

A total of 3000 patient files from the Ankara University Faculty of Medicine Multidisciplinary Behçet Outpatient Clinic, covering the period from 2010 to 2024, were evaluated. 300 patients were excluded because of duplicate records and 206 were excluded as they did not meet the International Criteria for Behçet's Disease (ICBD) (21). The files of patients who were diagnosed with BS and received MMF treatment were reviewed. Patients in whom MMF treatment was initiated for a reason other than vascular BS were excluded from the study. Twenty-six patients receiving MMF treatment were screened, and vascular involvement was found in 18 of them. 3 patients were excluded from the study due to extravascular BS, 1 patient due to renal transplantation, and 1 patient due to the initiation of MMF treatment for IgG4-related disease. 3 patients were excluded because they did not follow-up after MMF treatment was started. Patients who received MMF treatment for at least 6 months were included in the study. However, if there was drug discontinuation due to side effects in the first 6 months, this was also noted. Vascular involvement leading to MMF treatment was categorized into venous thrombus, pulmonary artery involvement (including aneurysm and/or thrombus), peripheral arterial involvement, and venous ulcer, with each noted individually. Due to the different course of venous ulcers, remission and relapse status were determined separately for venous ulcer and vein/artery involvement. Vascular remission is defined based on the lack of no new vascular events, and CRP levels below 10 mg/l. Vascular relapse is described by the development of a new vascular lesion or the continuation of an existing lesion despite the use of medication for ≥6 months of treatment. In the case of venous leg ulcers, we categorized the treatment response of patients into three groups: complete remission, partial remission, and relapse. We defined complete remission as the complete healing of ulcerated lesions, and partial remission as a reduction in both the number and/or size of these lesions, without the emergence of new ones. Relapse was defined by the development of new ulcers. The involvement of larger vessels was recorded in the patient records. We classified the pulmonary and peripheral arteries, the vena cava inferior (VCI)/vena cava superior, and the cardiac thrombus as larger vessels. Any extravascular involvement, such as neurological, ocular, or gastrointestinal, was also noted. We documented the immunosuppressive treatment for the same vascular condition before starting MMF treatment, along with the reasons for its discontinuation. The use of MMF treatment as induction or maintenance regimen and treatment related side effects were noted. Induction therapy describes the suppressive treatment administered during the initial 3-6 months following an acute vascular event, while

maintenance therapy is defined as the treatment provided after induction therapy. We recorded the CRP levels of patients prior to, at 6 months, and at 12 months of MMF treatment. Both vascular remission or relapse and extravascular relapse have been recorded at 12 months of treatment. Our primary endpoint was vascular remission at 12 months. The study was approved by the ethical committee of Ankara University Faculty of Medicine (İ10-799-24).

Statistics

We selected frequencies and percentages for categorical data. For numerical variables, we gave the results as median-interquartile range (IQR).

Result

A total of ten patients undergoing MMF treatment for vascular involvement were included in the study. Of the 10 patients, 90% were male. The median age at diagnosis was 23 (IQR 12) years, while the median age at the initiation of MMF was 32 (IQR 14) years. A total of 5 patients were initiated on MMF for deep vein thrombosis (DVT), 1 for pulmonary artery thrombus (PAT) and DVT, 2 for venous ulcers (One patient presented with both acute DVT and venous ulcer.), 1 for pulmonary artery aneurysm (PAA), and 1 for peripheral artery aneurysm (Table 1). Five patients underwent MMF treatment for remission induction, while another five patients received MMF for maintenance (Table 1). Patients used MMF for a mean duration of 25.1 (IQR 22.5) months. Relapses occurred in 2 out of 5 patients who were administered MMF as induction therapy, resulting in the development of acute DVT. Among the five patients receiving MMF as maintenance treatment, only one exhibited an active vascular event (new venous ulcer). Additionally, one patient was classified as vascularly active based on a CRP level of 10.3 mg/l; however, imaging findings could not confirm a new vascular event (Table 1). A patient with a history of erythema nodosum (EN) developed arthritis and EN following MMF treatment. Uveitis and DVT attack were observed in a patient with a history of uveitis under MMF treatment (Table 1). At 6 months, vascular remission was noted in 70% of patients, whereas at 12 months, it was noted in 60% of patients. MMF was discontinued in 1 patient at month 25 due to side effects (gastrointestinal intolerance), 2 patients had vascular relapse and 2 patients had leg ulcers and treatment was discontinued during follow-up. For the same indication, all of the patients received AZA treatment before MMF therapy. Of these, six discontinued azathioprine due to adverse effects, while vascular relapse occurred in the remaining four patients during azathioprine treatment (Table 1). Patients do not use interferon and anti-TNF agents before or in combination with MMF treatment. Anti-TNF treatment was started in 3 patients after MMF treatment was discontinued.

Eight patients showed involvement of larger vessels. When we looked at the history of larger vessel involvement, there were 3 VCI involvement, 4 PAT, 1 pulmonary artery aneurysm, 2 peripheral artery aneurysms and 1 intracardiac thrombus (Table 2). The history of extravascular involvement consisted of ocular involvement in four patients, brain parenchymal involvement in one patient, sinus vein thrombosis in two patients, and arthritis in three patients (Table 2). One of the 4 patients with a history of uveitis developed uveitis after MMF treatment, whereas the other 3 patients did not develop uveitis again. No relapses were observed in patients with neurological involvement receiving MMF treatment (Table 2).

The median CRP level of the patients prior to MMF treatment was 16.7 (IQR 51.8) mg/l. At the sixth month of treatment, the median CRP level was 5.7 (IQR 7.2) mg/l, and at the 12th month, the median CRP level is 7.5 (IQR 8.05) mg/l.

Discussion

This case series indicates that MMF therapy may be effective for both induction and maintenance treatment in VBS. Most patients who discontinued AZA treatment due to side effects in the first 12 months did not experience any MMF-related side effects. These findings indicate that MMF treatment may be an effective and safe option for patients with VBS. Vascular involvement in Behçet's syndrome is associated with significant morbidity and higher mortality rates (4, 22, 23). Thus, it is essential for patients with vascular involvement to receive effective and safe treatment. The 2018 EULAR guideline recommends use of immunosuppressive therapies and GCs for patients with VBS with venous involvement. Cases

Table 1. Characteristics of patients of during treatment with mycophenolate mofetil

	Type of Vascular Involvement Requiring MMF treatment	**Treatment given before Mycophenolate Mofetil treatment/Reason for discontinuation**	**Induction/ Maintenance/ Cumulative GCs Dose During MMF Treatment**	**Relapse/Remission (12. month)**	**Extravascular relapse (12. Months)**	**Reason for MMF discontinuation**
Patient 1	Chronic Leg Ulcer	Azathioprine, Gastrointestinal intolerance	Induction /600mg	Partial Remission		
Patient 2	Pulmonary Thrombus+ Deep Vein Thrombosis	Azathioprine, Gastrointestinal intolerance	Induction/3720 mg	Remission		
Patient 3	Deep Vein Thrombosis	Azathioprine, Development of Deep vein thrombosis and arthralgia	Induction/4000 mg	Relapse (Deep Vein Thrombosis)	Uveitis	Activation of vascular and ocular disease
Patient 4	Deep Vein Thrombosis	Azathioprine, Elevated liver function tests	Maintenance/2200 mg	Remission		After 38 months, development of erythema nodosum, arthritis and leg ulcers
Patient 5	Chronic Leg Ulcer+ Deep Vein Thrombosis	Azathioprine, New ulcer development	Maintenance/1800 mg	Relapse (New ulcer)		Development of new ulcers
Patient 6	Deep Vein Thrombosis	Azathioprine, Development of Deep vein thrombosis	Induction /1000mg	Remission		
Patient 7	Pulmonary Artery Aneurysm	Cyclophosphamide treatment for induction, azathioprine treatment for maintenance, azathioprine is discontinued due to leucopenia	Maintenance /0mg	(CRP:10.3 mg/l, not new event)		
Patient 8	Peripheral Artery Aneurysm	Azathioprine, Gastrointestinal intolerance	Maintenance/0 mg	Remission		GIS intolerans at 25th month
Patient 9	Deep Vein Thrombosis	Azathioprine, leucopenia	Maintenance/2000 mg	Remission		
Patient 10	Deep Vein Thrombosis	Azathioprine, Gastrointestinal intolerance, Deep Vein thrombosis	Induction /0 mg	Relapse (Deep Vein thrombosis)		Activation of vascular disease

Table 2. History of VBS patients with larger vessel and extravascular organ involvement

Patient	Type of Larger Vessel	Chronological order of vascular events	All immunosuppressants used prior to mycophenolate mofetil	Neurological Involvement (and Type)	Ocular Involvement	Arthritis
Patient 1	Vena Cava Inferior	DVT→VCI → DVT→PAT→Leg ulcer	Cyclophosphamide and azathioprine	None	None	None
Patient 2	Vena Cava Inferior +Pulmonary Artery Thrombus	VCI→PAT+DVT	Azathioprine	None	None	None
Patient 3	Pulmonary Artery Thrombus	DVT→PAT→→DVT	Cyclophosphamide and azathioprine	Yes (Cerebral Sinus Vein Thrombus)	Yes	Yes
Patient 4	None	DVT	Azathioprine	None	Yes	None
Patient 5	Vena Cava Inferior	VCI and DVT are detected in the chronic phase. It is not known which one first.	Azathioprine	None	None	Yes
Patient 6	None	DVT	Azathioprine	Yes (Cerebral Sinus Vein Thrombus	None	None
Patient 7	Pulmonary Artery Aneurysm and Thrombus	PAA+PAT	Cyclophosphamide and azathioprine	None	None	None
Patient 8	Peripheral Artery Aneurysm	DVT→ Peripheral Artery Aneursym	Azathioprine	None	Yes	Yes
Patient 9	Pulmonary Artery Thrombus +Intracardiac thrombus	Pulmonary artery thrombus + intracardiac thrombus simultaneously	Cyclophosphamide and azathioprine	Yes (Parenchymal)	None	None
Patient 10	Peripheral Artery Aneurysm	Peripheral Artery Aneurysm→DVT	Interferon, cyclophosphamide and azathioprine	None	Yes	None

Abbreviations: DVT: Deep Vein Thrombosis, VCI: Vena Cava Inferior, PAT: Pulmonary Artery Thrombus, PAA: Pulmonary Artery Aneurysm

of resistance indicate the use of monoclonal TNFi. In individuals with arterial involvement, it is recommended to start therapy with high-dose GCs and cyclophosphamide or monoclonal TNFi (13). AZA use is known for its capacity to decrease the probability of vascular events (17). There are no prospective randomized controlled trials on AZA treatment in patients with VBS. A study that involved a small group of patients with venous thrombosis demonstrated that CsA treatment effectively prevents relapse and the progression of venous insufficiency (24). However, the presence of many side effects of CsA such as hypertension and nephrotoxicity limit its use (25). Monoclonal TNFi therapy is a highly effective treatment in patients with VBS. This has also been shown by randomised controlled and retrospective trials. Increased tuberculosis risk has been observed in BS patients receiving TNFi treatment (26-30). Some patients avoid the use of TNFi because of its side effects. If AZA-related side effects are observed in this patient group, there is a need for immunosuppressive agents that can be used long-term. However, no retrospective or prospective study showing the efficacy of MMF treatment in VBS patients has been found in the literature. MMF treatment

can be helpful in different kinds of vasculitis. Data regarding the usage of MMF in BS are notably scarce. In the literature, GCs and MMF treatment was reported to be effective in a pediatric patient with BS with cerebral sinus vein thrombosis (19). There are studies showing that MMF treatment is effective in Neuro-BS patients (18). A study involving BS patients with ocular involvement showed that MMF treatment can be administered alongside monoclonal TNFi and may be used for maintenance therapy (20). The data regarding mucocutaneous involvement are controversial. When we look at the literature, there is a study showing that MMF medication reduces mucocutaneous findings as well as another study showing that it increases them (31, 32). In our patient series, MMF treatment was given for vascular involvement in 10 patients. Over a 12-month period, new deep vein thrombosis (DVT) was detected in only two patients, and one patient developed a new leg ulcer. Among the group undergoing maintenance therapy with MMF, new ulcer development occurred in only one patient at the 12-month, and no cases of vascular event was noted. This finding suggests that MMF treatment may be effective in induction and maintenance therapy, especially maintenance therapy, in patients with VBS. T cells, antigen-presenting cells, and neutrophils are involved in the pathogenesis of BS. Activated neutrophils result in recruitment of neutrophils and lymphocytes to the activated area (33). MMF is an agent that prevents the recruitment of these cells to the site of inflammation by acting on T and B lymphocytes (34). The effect of MMF treatment on lymphocytes suggests that it can be used to treat patients with VBS. In our case series, elevated liver function tests noted during azathioprine treatment were absent during MMF treatment. Patients who discontinue AZA treatment due to adverse effects could use MMF as an acceptable alternative for induction or maintenance therapy. The small number of patients, retrospective collection of the data, and lack of comparison with other immunosuppressive therapies are important limitations of our study. In conclusion, MMF therapy may be an effective and safe treatment agent that can be preferred over azathioprine and cyclosporine A in VBS patients, especially in maintenance therapy, which has been overlooked for years. Our study is important because it suggests for the first time that MMF treatment may be effective in VBS patients. Further large-scale prospective studies are needed to support the efficacy of MMF in VBS.

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